Modeling Mechanical Cardiopulmonary Interactions for Virtual Environments*

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We have developed a computer system for modeling mechanical cardiopulmonary behavior in an interactive, 3D virtual environment. The system consists of a compact, scalar description of cardiopulmonary mechanics, with an emphasis on respiratory mechanics, that drives deformable 3D anatomy to simulate mechanical behaviors of and interactions between physiological systems. Such an environment can be used to facilitate exploration of cardiopulmonary physiology, particularly in situations that are difficult to reproduce clinically. We integrate 3D deformable body dynamics with new, formal models of (scalar) cardiorespiratory physiology, associating the scalar physiological variables and parameters with corresponding 3D anatomy. Our approach is amenable to modeling patient–specific circumstances in two ways. First, using CT scan data, we apply semi–automatic methods for extracting and reconstructing the anatomy to use in our simulations. Second, our scalar models are defined in terms of clinically–measurable, patient–specific parameters. This paper describes our approach and presents a sample of results showing normal breathing and acute effects of pneumothoraces.

Introduction

There is a growing sense that the computer will be the tool that will revolutionize learning. Nowhere more is this felt than with the promise of 'virtual reality,' simulated environments in which users interact with virtual objects as one would interact in real life. In medicine, this will mean the computer can help students study anatomy and physiology, or practice surgical procedures on simulated patients. A virtual environment can enable students to visualize and explore scenarios that are difficult to reproduce or otherwise impossible to view in clinical settings. However, the current generation of graphical virtual environments tend to focus on the visual presentation, especially surgical simulation, with less emphasis on rigorous physiological modeling. But as we allow students more freedom to explore, the simulations will require more robust models of physiology to predict outcomes accurately.

In the modeling of deformable tissue, we need to be concerned both with acceptable visual feedback and correct modeling of the physiological effects of the deformations. Forces that produce deformations are either produced by the user (through interaction with the body) or developed internally from the tissue. These forces can influence not only the shape of the object but also the physiological processes that involve it. This means we need models of *mechanics*. Mechanics describes the relationship between forces, displacements, and their derivatives. For example, if one squeezes or clamps a vessel through which fluid flows, resistance to flow within that vessel increases. If the vessel is part of a larger network, the increased resistance can propagate effects throughout the system.

Ideally, one could define the mechanical characteristics of systems so that arranging them as they are in the body would produce the real mechanical interactions that result. The problem is that real 3D deformable body mechanics is computationally exorbitant and requires such detailed measurements that would prohibit adapting the system to individual patients.

With the goals of producing correct mechanical behavior within a rich a virtual environment for medical education, we have defined a compact, scalar description of cardiopulmonary mechanics, with an emphasis on respiratory mechanics, that drives deformable 3D anatomy to simulate mechanical behaviors of and interactions between physiological systems. The scalar description provides an efficient form with sufficient degrees of freedom to express a range of normal and pathological conditions, including acute effects of pneumothoraces. By its definition, the scalar description contains clinically–measurable, patient–specific parameters. The 3D anatomy, reconstructed from CT scans, provides a vivid depiction of the simulations as well as a natural means for interacting with the patient. We couple these methods by translating (i) scalar predictions to the 3D anatomy and (ii) 3D deformation to its impact on scalar physiological properties. The program allows the user to

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interact with the virtual patient by changing mechanical parameters or introducing injuries such as a breach in the chest wall.

Review of Anatomy and Physiology

Ventilation is the process by which air moves into and out of the lungs. The lungs are contained within the thoracic cavity which is divided into two halves (one for each lung), separated by the mediastinum. The mediastinum is a membranous structure that contains the heart, the major blood vessels, and the trachea. Clinically, the rib cage and diaphragm/abdomen are referred to as the 'chest wall,' a unit comprising the ribs, sternum, respiratory muscles, diaphragm, abdominal contents, and abdominal muscles.

The lung and chest wall are separated by a thin layer of liquid called the intrapleural fluid occupying the intrapleural space. Normally, the fluid is at a subatmospheric pressure. The intrapleural fluid allows the lung to slide on the chest wall and acts to transmit forces between them. Air or blood entering the intrapleural space can cause the lung to separate from the chest wall and impede respiration. A *pneumothorax* is the pathological presence of air in the intrapleural space, caused for example, by a breach in the chest wall. If the breach allows the free bidirectional passage of air, it can be called an *open sucking chest wound.* If a flap of tissue at the breach acts as a one–way valve, allowing air into the intrapleural space but plugging the hole to prevent air from leaving during expiration, pressure builds up and a *tension pneumothorax* develops. Intrapleural pressure increases in the half of the thoracic cavity in which the pneumothorax occurs. The resulting pressure gradient across the mediastinum pushes the mediastinal contents towards the opposite side.

Blood flow is essential for delivering nutrients and oxygen to body tissue and removing the waste products of metabolism. Blood is pumped by the heart into the pulmonary and systemic circulation. The right ventricle of the heart pumps blood into the pulmonary circulation (through the lungs), and the left ventricle pumps it into the systemic circulation (rest of the body). The blood returning to the heart from the systemic circulation arrives via the inferior and superior vena cavae. The heart and vena cavae are located in the mediastinum.

Mechanical Interaction. Because the vena cavae are flaccid and the blood pressure within very low, they can be greatly influenced by changes in the pressure outside their walls, which we call the intramediastinal pressure. Changes in vessel dimensions alter flow resistance properties. Assuming that intramediastinal pressure changes as the average of the left and right intrapleural pressures, one can see that intrapleural pressure changes, originating from ventilation, may cause changes to cardiovascular function. The tension pneumothorax and severe open sucking chest wounds are classic examples. With pneumothoraces, the increasing intrapleural pressure may result in collapse of the vena cavae, with immediate, life-threatening implications. This is just one example of mechanical imbalance between the left and right halves of the thoracic cavity affecting the mediastinal contents.

Background

The current generation of anatomical atlases, such as the many efforts involving the Visible Human Project [1], use virtual environments to study and dissect realistic 3D anatomy. However, any 'physiology' in these programs means labels, text, or canned animations. Recently, there has been great interest in simulating endoscopic procedures which require dynamic simulation of soft tissue. These programs are mostly developed for very specific procedures and do not let the user explore consequences of incorrect actions. With today's increasing computational power, the medical graphics community is exploiting developments in physics–based, deformable body dynamics for modeling soft tissue. Cover et al. [2] discuss various approaches in the literature and their limitations, and propose a novel method based on energy–minimizing splines.

There are a number of comprehensive body simulation programs developed from a traditional bioengineering perspective, with current commercial products mainly for anesthesia training [4]. These approaches rely on sophisticated mathematical models for physiological systems. However, the visualization of anatomy is relatively simple, such as changing skin color or using video clips. Because they are designed for anesthesia training, these models tend to center on pharmacokinetics (absorption, metabolism, and action of drugs) and pharmacodynamics (drug effects on patients) with less stress on complexities of physical movement. Generally, these systems use the simplest chest wall mechanics model, one in which the contributions of the rib cage and diaphragm/abdomen combination act with a single degree of freedom. The problem with this is that one cannot investigate the effects of different muscle recruitments or anomalies within the chest wall. In contrast, our approach emphasizes mechanics. When we build and integrate other physiological components, incorporating the influences of neurology and biochemistry, we can exploit mechanical novelties to capture a wider range of physiological phenomena more realistically.

Scalar Physiological Modeling

Respiratory Mechanics. Our ventilation model extends the work of Primiano [5]. The process of developing a model involves deciding on the significant degrees–of–freedom for the system being modeled, describing the relationship between components using mechanical elements, and finally writing force– and volume–balance equations that express those relationships. Figure 1 is an example physical analog to the ventilatory anatomy. It contains two lungs, the mediastinum, the rib cage, the diaphragm, the abdomen, and other parts which are fully explained in [3].

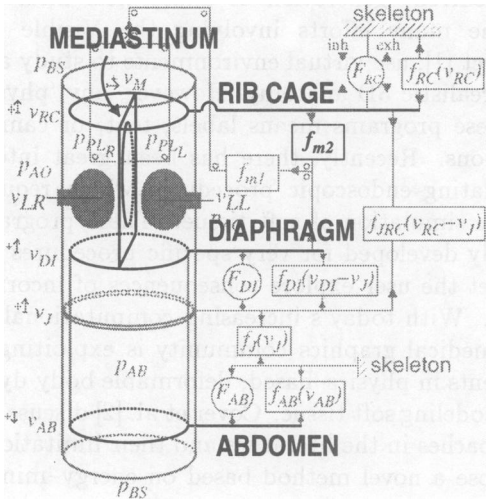

Figure 1: Example physical analog.

In the physical analog, the chest wall components are represented as (massless) disks that move within a conceptual cylinder. The uppercase F (within circles) represent active muscular efforts, while the lowercase f (within squares) represent passive mechanical elements. The arrows indicate the direction of influence an element has on a component. For example, tension in the mediastinal element f_{m_2} resists the rib cage's positive (upward) displacement. We use the direction of the arrows to determine the signs, when the pieces are written in equation form. The tension in an element depends on the relative displacement of the named component, with zero being its value at the patient's normal rest. The diaphragm and abdomen move with a single degree of freedom because we model the abdomen as a liquid of uniform pressure. As a first approximation, we model the mediastinum and its contents computationally as an elastic membrane.

Figure 1 is just for illustration. Our actual model has individual left and right rib cage and diaphragm components, as well as bilateral intrapleural spaces of non–zero volume changes to model pneumothoraces. We currently model the passive elements as viscoelastic. This means that an element x influences the change in volume v_y by the function $f_x(v_y) = v_y/C_x + \dot{v}_y R_x$, where C_x is the element's compliance and R_x is the element's resistance. The result of applying this process is a system of ordinary differential equations (see [3] for details) which we solve during the simulation explicitly over time using conventional numerical methods.

Cardiovascular Mechanics. To simulate basic cardiovascular mechanics, we adapted Rideout's `PF-1` model [6] which incorporates linear and non–linear (time–varying ventricular–compliance) elements. We call the portion of the systemic venous circulation closest to the right atrium the *vena cava*, a collective representation of the inferior and superior vena cavae. In Rideout's model, the pressure outside a vessel is assumed not to change. In our model, we identify a certain section of the vasculature as being 'within' the mediastinum and hence influenced by changes in the pressure outside the vessels. To these vessels we apply the transmural pressure as the difference between the pressure within the vessel and the change in intramediastinal pressure (equal to the average of the changes of the two intrapleural pressures).

3D Anatomical Modeling

The 3D anatomy in our environment consists of two lungs, an upper torso, the rib cage (from Viewpoint DataLabs), the parietal pleura (encasing each lung), and the inferior and superior vena cavae (IVC and SVC, respectively). We divide the parietal pleura into its medial surface, corresponding to a lateral wall of the mediastinum, the inferior surface, corresponding to the diaphragm, and the rib cage. We do not currently have a 3D representation for the abdomen.

In the lung/chest wall system, the rib cage muscles, diaphragm, and abdominal muscles provide the active, driving forces for displacements, while the lungs, mediastinum, and mediastinal contents are passive. We have divided the modeling of structures into two categories: structures controlled by 3D kinematics and structures controlled by 3D dynamics. Because computation speed is a great concern, we cannot use the latter to model everything. It is important to model the lungs with deformable–body dynamics because they must be able to follow and slide on the chest wall, as well as collapsing in ways that may be difficult to model realistically with pure kinematics. The structures with kinematics are updated with linear interpolation. The structures with dynamics are viscoelastic spring–mass systems, updated with the Newtonian equation of motion $\sum f = ma$ (described

in [3]). The surfaces of the IVC and SVC are modeled as deformable meshes. The rib cage, diaphragm, and mediastinum are modeled with kinematics. Their displacements are interpolated based on the values of their associated pressure and volume variables.

For small lung volume changes about an operating point (e.g., tidal breathing), the lung acts like an isotropic solid. This is true regardless of the operating point [7]. For large volume changes, the lung cannot be modeled as an isotropic solid, because large changes involve changes in airway dimensions as well as alveoli expansion.

To capture the isotropic and anisotropic changes efficiently, we model each lung with a mixed dynamics/kinematics process. The anisotropic 'core' of the lung gives the basic shape and is updated with kinematics (linear interpolation) based on the transmural pressure gradient (the difference of alveolar and pleural pressure changes). The surface of the lung is modeled as a deformable mesh connected to the core by dampened springs. Forces are applied proportional to the transmural pressure along surface normals, producing isotropic change. The parts controlled by dynamics are updated with respect to body forces, contact with other structures, internal forces arising from the passive elements in the structures trying to return them to their unstressed shapes, and pressure gradients developed in the scalar modeling.

We estimated the mechanical properties based on reconstructed anatomy from CT scans of a subject breathing different volumes of air. We obtained sets of CT scans of the thorax for a subject breathing different volumes of air, courtesy of Dr. Eric Hoffman at the University of Iowa. The reconstructed versions of these data sets became the boundary conditions for our 3D simulation. The process from CT scans to 3D geometry is described fully in [3].

Scalar and 3D Integration

The 3D and scalar models are coupled during the simulation in two ways. First, the kinematics parts are updated with linear interpolation based on pressure gradients or volumes predicted in the scalar models. Second, the dynamics parts are sensitive to 3D forces derived from pressure gradients. We convert the scalar transmural pressure to 3D forces acting along surface normals. These 3D forces act to deform the anatomy. Collisions between the lung and the chest wall are resolved by allowing the lung to slide along the wall, but not penetrate it. The walls of the mediastinum are attached to the contents (currently just the vena cavae) by dampened springs. This means that mediastinal deformations may cause vessel deformations. Depending on the degree of vessel deformation (judged by changes to the cross–sectional area), the program changes the vessel's flow resistance (since the flow resistance is inversely proportional to its cross–sectional area).

To keep the scalar and 3D models consistent, we must ensure that a pressure gradient applied to the 3D model results in a volume consistent with the scalar volume predicted. This is a known problem with spring–mass systems which we will address in the future by introducing volume–preserving forces.

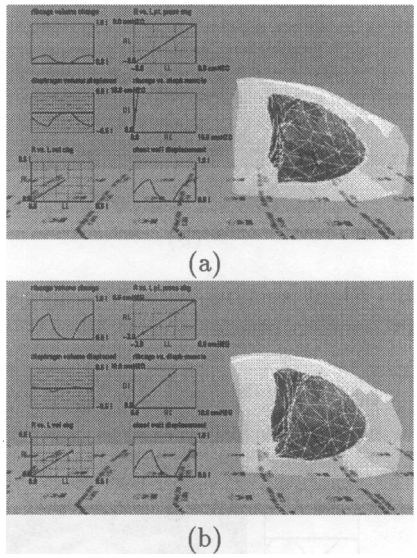

Figure 2: Results from increasing rib cage muscle effort relative to diaphragmatic muscle effort. We show in (a) the normal tidal expansion, and (b) the expansion with an increased rib cage muscle force.

Results

The system gives the user the choice of displaying over seventy scalar variables plotted against time or each other, as well as control of over seventy parameters. In our implementation, the parameters are resistances, compliances, and coefficients for muscle influences. We chose initial parameter values to produce normal responses for a healthy individual. The program lets the user orient the view arbitrarily in the 3D environment. Figure 2 shows a view of the (supine) patient from the patient's left lateral position. The prints below the anatomy are on a virtual bed sheet. The bed reminds the user of the orientation regardless of the user's viewing perspective.

In severe pneumothoraces, such as depicted in the right thoracic cavity in Fig. 3, the intrapleural pressure can become equivalent or greater than atmospheric pressure, causing the right lung to collapse and the mediastinum to shift markedly. This has a pronounced,

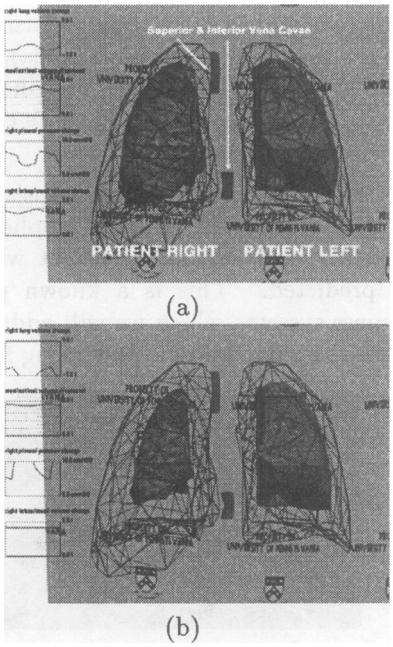

Figure 3: Right sucking chest wound (a) compared with right tension pneumothorax (b).

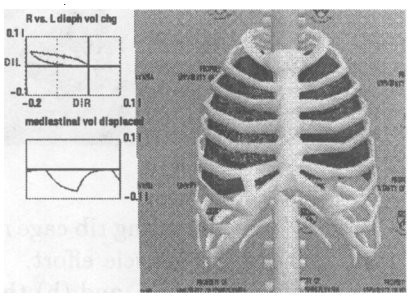

Figure 4: Results of paralyzing the left diaphragm. Left and right diaphragm displacements are no longer equal and the mediastinum is pushed into the right side of the chest.

detrimental effect on venous return because of cardiopulmonary mechanical interactions. Figure 3 shows the different results from a right open sucking chest wound (Fig. 3(a)) and a right tension pneumothorax (Fig. 3(b)). The right lung is appreciably smaller in the tension pneumothorax because the intrapleural pressure is greater. Because we have not yet implemented a volume preservation scheme, the left lung separates from the chest wall.

In the simulated tension pneumothorax of Fig. 3(b), the blood pressure decreases significantly. Because the flow resistance in the vena cava has increased, the central venous pressure and the central venous volume increase. It is important to remember that these results reflect only the mechanical consequences of the injuries. More realistic clinical presentations can be obtained with the incorporation of neurological models.

In Fig. 4, we show the result of paralyzing the left diaphragm. The plots displayed are the right (x–axis) versus left diaphragm (y–axis) displacement (top), and the displacement of the mediastinum. The plots reveal that the left diaphragm is being sucked into the left chest (positive displacement is upward). Also of interest is that the sides of the diaphragm no longer expand and contract by the same amount and at the same time–the change we made has the effect of making diaphragmatic behavior depend on its history (called 'hysteresis'). The plot below this reports that the mediastinum is drawn into the right side of the thoracic cavity, approximately 80 ml.

Conclusion

We have proposed the development of a general framework for simulating mechanical behavior in a virtual environment and suggested its potential value in creating useful programs for medical education. We are optimistic about the benefits that virtual environments will provide in medical education because they have the potential to provide an engaging experience which we believe is certain to enhance the learning process.

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